More perinatal deaths associated with poor long-term variability during antenatal fetal heart-rate monitoring

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Summary

Positive stress and non-stress tests of 243 infants were examined for accelerations and the duration of poor long-term variability during fetal heart rate monitoring. Accelerations were present in 47% when the variability was good. No accelerations were seen when poor variability lasted for more than 75% of the monitoring time; this was also associated with lower birth-weights, shorter gestational duration and lower 5-minute Apgar scores. These measurements improved as the period of poor variability decreased. Intra-uterine death occurred in 1,9% of infants when the variability was good, in 3,6% when the variability lasted for less than 75% of the recording time and in 19,6% when the poor variability lasted longer than 75% of the recording time. Neonatal deaths occurred in 7,5%, 11,5% and 21,7% of these three groups, respectively. Poor long-term variability was also associated with growth-retardation.

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Non-stress tests sometimes fail to give an indication of the true fetal condition, since a non-reactive pattern may represent either fetal sleep or fetal compromise.[1] Non-reactive non-stress tests also occur more frequently in preterm pregnancies.[2,3] In 16% of mothers nipple stimulation fails to produce adequate contractions,[4] especially in the preterm pregnancy where the uterus is not yet responsive to oxytocic stimulation. Hyperstimulation occurs in about 2,8% of mothers in whom nipple stimulation has been applied. This could occasionally cause fetal bradycardia.[5] Although it has been demonstrated that hyperstimulation, as seen during contraction stress tests, is not harmful to the fetus, excessive uterine contractions could be dangerous to the compromised fetus. Pregnant patients with severe preterm proteinuric hypertension create a special problem because of the higher prevalence of non-reactive tests and underlying placental insufficiency,[6] which add to the risks of hyperstimulation. Since 36% of intra-uterine deaths in patients with severe proteinuric hypertension are due to abruptio placentae,[7] it would be desirable to monitor the fetal heart rate at frequent intervals in such patients. For the above reasons it would not be appropriate to perform stress tests for all non-reactive tests. It would also be time-consuming to draw up a biophysical profile after each non-reactive non-stress test. Other parameters of the fetal heart rate pattern, such as long-term baseline variability, should therefore be assessed for clinical purposes.

A retrospective study was undertaken on 262 positive stress and non-stress tests performed between February 1975 and January 1987.

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Patients and methods

During the performance of the test the usual precautions were taken to reduce the possibility of supine hypotension. Repeated late decelerations were regarded as indications for delivery. After the first 102 tests, it was found that neonatal survival in babies weighing < 1 000 g was very poor.[8] It was therefore decided not to deliver babies with an estimated weight < 1 000 g. However, as the large majority of these infants subsequently suffered intra-uterine death and as neonatal intensive care facilities had in the meantime improved, it was decided to deliver all patients when the fetal heart rate pattern showed repeated late decelerations unless the gestational age was less than 28 weeks or unless the calculated weight was < 800 g. Maternal neonatal data were collected prospectively and filed with the fetal heart rate recordings.

For the present study the fetal heart rate tracings of 262 infants were re-examined for late and variable decelerations, accelerations and long-term variability. Late decelerations were defined as a decline in the fetal heart rate of at least 10/min, starting at least 30 seconds after the contraction and lasting for at least 60 seconds. When contractions were not shown on the tracing, decelerations were regarded as late if there was a gradual decline in fetal heart rate followed by a slow return to the basal rate. Long-term variability was regarded as reduced when it was less than 5/min for at least 5 minutes (Fig. 1). The duration of the reduced variability was also calculated and expressed as a percentage of the duration of the test. Variability of more than 5/min for longer than 5 minutes was regarded as good (Fig. 2).

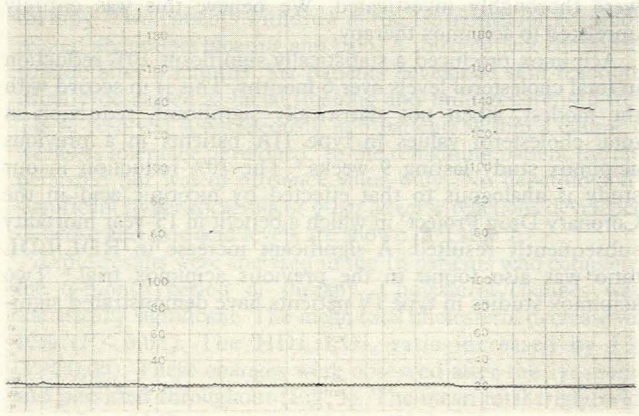

Fig. 1. Reduced long-term variability.

Finally, patients were categorised according to the presence and duration of reduced long-term variability. These two groups were then compared with reference to acceleration patterns, late decelerations, fetal distress during labour, intra-uterine death, neonatal death, gestational age, birth weight and growth retardation. The latter was assessed by the growth curves of Dunn.[9] In the case of continuous measurements the distribution of the measurements was firstly assessed, and

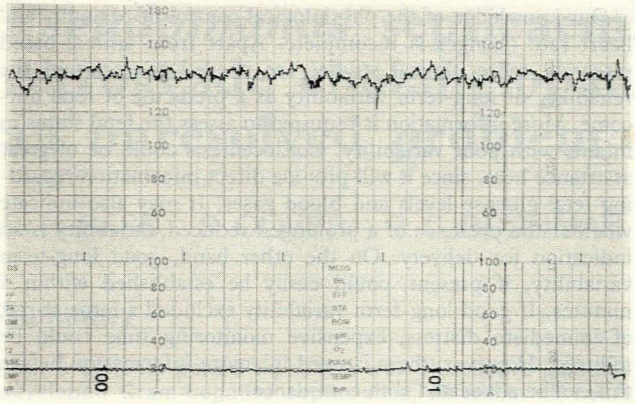

Fig. 2. Normal long-term variability.

then, if the distribution was normal, the *t*-test was used to compare the data. A *P* value of < 0,05 was accepted as statistically significant.

Results

Since information on 19 infants was incomplete or there were twin pregnancies, the data on 243 infants were analysed. Indications for the tests were as follows: pre-eclampsia 129 (53%), suspected growth retardation 57 (23%), post-term pregnancy 25 (10%), miscellaneous 13 (5%), diabetes 10 (4%), and antepartum haemorrhage 9 (4%). Duration of the tests ranged from 9 minutes to 446 minutes (mean 49 minutes). Good long-term variability was seen in 112 (46%) infants and accelerations were present in 53 (47%) of them. Reduced long-term variability, which lasted for < 25% of the recording time, was found in 36 (15%) infants; 8 (22%) also had acceleration patterns. In 35 infants (14%) the reduced variability lasted between 25% and 50% of the recording time; 6 (17%) also demonstrated accelerations. Only 13 infants (5%) demonstrated reduced variability which lasted 50 - 75% of the recording time. Only 1 recording also demonstrated accelerations. Reduced long-term variability lasted > 75% of the recording time in 47 infants (19%); no accelerations were found here.

Since the gestational ages, birth weights and 5-minute Apgar scores of infants in whom the reduced variability lasted for 1 - 25%, 25 - 50% and 50 - 75% of the recording time, respectively, did not differ, they were put into the same group. Three groups were, therefore, compared: (*i*) those infants with no reduced long-term variability: (*ii*) those in whom the reduced variability lasted less than 75% of the recording time; and (*iii*) those in whom reduced variability lasted longer than 75% of the recording time.

The mean duration of pregnancy was 37,5, 35,0 and 32,7 weeks, respectively (Table I). Mean birth-weights for the three groups were 2 375 g, 1 920 g and 1 359 g, respectively. The 5-minute Apgar scores for these groups were 8,5, 7,4 and 5,4, respectively.

Intra-uterine death occurred in 1,9% of infants in whom there was no reduced variability, in 3,6% in whom the reduced variability was < 75% and in 19,6% in whom the reduced variability was > 75% of the recording time. For neonatal deaths these figures were 7,5%, 11,5% and 21,7%, respectively (Table II). Information on the neonatal outcome of 6 of the infants could not be found. When the fetal heart rate variability was good, intra-uterine growth retardation occurred in 39% of cases but it increased to 47% and 60% when poor variability was present for < 75% and 75 - 100% of the recording time, respectively.

The two intra-uterine deaths which occurred when the variability was good were due to abruptio placentae (1 infant weighing 800 g) and placental insufficiency (2 500 g). There were 3 intra-uterine deaths in the group of infants in whom poor variability was present for < 75% of the recording time; 1 was the result of abruptio placentae (infant weighing 1 200 g) and the other 2 were due to placental insufficiency (880 g and 920 g). In the group of infants in whom poor variability was present for 75 - 100% of the time, 7 fetuses were regarded as too small to be delivered. All weighed ≤ 1 000 g. The remaining 2 fetuses in this group weighed > 1 000 g; 1 weighing 1 800 g was not delivered because of a poor lecithin/sphingomyelin ratio and 1 of 1 500 g demonstrated a terminal fetal

TABLE I. CORRELATION BETWEEN DEGREE OF FETAL HEART RATE VARIABILITY AND OUTCOME

	Good variability	Poor variability (< 75%)	Poor variability (75 - 100%)	Significant differences ($P < 0,05$)
No. of patients	**112**	**84**	**47**	
Acceleration patterns	**53 (47%)**	**15 (18%)**	**0**	
Gestational age (wks)	**37,5 ± 4,5**	**35,0 ± 4,5**	**31,7 ± 3,9**	**Between all three**
Birth-weight (g)	**2 375 ± 886**	**1 920 ± 792**	**1 359 ± 68**	**Between all three**
5-minute Apgar score	**8,5 ± 2,2**	**7,4 ± 2,8**	**5,4 ± 3,6**	**Between all three**

TABLE II. CORRELATION BETWEEN POOR FETAL HEART RATE VARIABILITY AND PERINATAL MORTALITY

	Good variability		Poor variability (< 75%)		Poor variability (75 - 100%)		
	No.	%	No.	%	No.	%	Total
Intra-uterine death	**2**	**1,9**	**3**	**3,6**	**9**	**19,6**	**14**
Neonatal death	**8**	**7,5**	**10**	**11,9**	**10**	**21,7**	**28**
Survivors	**98**	**90,6**	**71**	**84,5**	**27**	**58,7**	**195**
Total	**107**		**84**		**46**		**237**

heart rate pattern at the first test, and died before a caesarean section could be done.

Causes of the 8 neonatal deaths in the normal variability group were congenital abnormalities (3), hyaline membrane disease (2), intraventricular haemorrhage (2), and septicaemia (1). In the < 75% poor variability group the 10 neonatal deaths were caused by hyaline membrane disease (5), asphyxia (2), congenital abnormalities (1), pulmonary haemorrhage (1), and necrotising enterocolitis (1). In the 75 - 100% poor variability group the 10 neonatal deaths were due to hyaline membrane disease (4), asphyxia (2), necrotising enterocolitis (1), gastroenteritis (1), massive pulmonary haemorrhage (1) and a blocked endotracheal tube on day 22 (1).

Discussion

During this study accelerations were found in 46% of infants with a good fetal heart rate variability. If it is considered that the duration of the antenatal monitoring ranged from 9 minutes to 446 minutes with a mean of 49 minutes, it can be assumed that more accelerations would have occurred in the group with good variability if the period of monitoring had been increased. (It has been found that quiet periods of the fetus have a duration of 12 - 93 minutes.[10]) Since the presence of accelerations reflects fetal well-being,[11] its presence in infants with good long-term variability indicates a favourable outcome. No accelerations were found in the infants in whom poor variability lasted for > 75% of the recording time. This finding could be partly explained by the fact that the gestational age was less in this group because it has been demonstrated that reactivity of the fetal heart rate improves with advancing gestational age.[3,12]

Infants with poor long-term variability also demonstrated a poor fetal outcome as judged by the 5-minute Apgar score and intra-uterine and neonatal deaths. Prematurity certainly played a role in the lower 5-minute Apgar scores and the increased neonatal deaths in the infants with poor variability, but the greater number of intra-uterine deaths in this group cannot be ascribed to the difference in gestational age. Poor long-term variability therefore carries a higher risk of intra-uterine death. A cause for concern is the intra-uterine death, possibly owing to placental insufficiency, in the group with good variability. It demonstrated that good variability does not completely exclude the possibility of intra-uterine death. Intra-uterine growth retardation occurred significantly more often in the infants with poor variability. This could be due to acidaemia or a general reduction in fetal activity, as has recently been demonstrated.[13,14]

Our knowledge of the physiological control of periodic fetal heart rate changes is incomplete. Apart from animal-based research, more clinical studies are needed to ascertain the reliability of long-term variability as a predictor of fetal outcome. More information is required to assess the fetal status in infants with poor variability. Cordocentesis could be of great assistance here, since it will provide direct information regarding fetal glucose levels and blood gases. It may also indicate whether the presence of a prolonged reduced variability is an indication for delivery. On the other hand, good long-term variability, if present, could easily be established within 5 minutes. If good long-term variability excluded a fetus at risk of immediate distress, expensive monitoring time could be reduced. However, there is need for more information before this can be advocated with confidence.

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